WEST Search History

DATE: Tuesday, April 29, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L7	L6 and (l2 or l1)	50	L7
L6	L5 and l3	535	L6
L5	antisigma$ or anti-sigma$ or sigma$ or sigma28$ or anti-sigma28$ or anti-sigma28$	82380	L5
L4	antisigma$ or anti-sigma$ or sigma$	82380	L4
L3	helicobacter or pylori	1632	L3
L2	(((435/252.3)!.CCLS.))	6103	L2
L1	((435/232)!.CCLS.)	368	L1

END OF SEARCH HISTORY

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
****
ENTER PASSWORD:
****
Welcome to DIALOG

Dialog level 02.12.60D

Last logoff: 17apr03 16:14:39
Logon file405 22apr03 12:09:53
*** ANNOUNCEMENT ***
***
--File 515 D&B Dun's Electronic Business Directory is now online
completely updated and redesigned. For details, see HELP NEWS 515.
***
--File 990 - NewsRoom now contains October 2002 to present records.
File 993 - NewsRoom archive contains 2002 records from January 2002-
September 2002. To search all 2002 records, BEGIN 990,993 or B NEWS2002
***
--Alerts have been enhanced to allow a single Alert profile to be
stored and run against multiple files. Duplicate removal is available
across files and for up to 12 months. The Alert may be run according
to the file's update frequency or according to a custom
calendar-based schedule. There are no additional prices for these
enhanced features. See HELP ALERT for more information.
***
--U.S. Patents Fulltext (File 654) has been redesigned with
new search and display features. See HELP NEWS 654 for
information.
***
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
***
--CLAIMS/US Patents (Files 340,341, 942) have been enhanced
with both application and grant publication level in a
single record. See HELP NEWS 340 for information.
***
--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
***
--Important news for public and academic
libraries. See HELP LIBRARY for more information.
***
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
***
NEW FILES RELEASED
***Dialog NewsRoom - Current 3-4 months (File 990)
***Dialog NewsRoom - 2002 Archive (File 993)
***Dialog NewsRoom - 2001 Archive (File 994)
***Dialog NewsRoom - 2000 Archive (File 995)
***TRADEMARKSCAN-Finland (File 679)

***TRADEMARKSCAN-Norway (File 678)
***TRADEMARKSCAN-Sweden (File 675)
***
UPDATING RESUMED
***Delphes European Business (File 481)
***
RELOADED
***D&B Dun's Electronic Business Directory (File 515)
***U.S. Patents Fulltext 1976-current (File 654)
***Population Demographics (File 581)
***Kompass Western Europe (File 590)
***D&B - Dun's Market Identifiers (File 516)

REMOVED
***Chicago Tribune (File 632)
***Fort Lauderdale Sun Sentinel (File 497)
***The Orlando Sentinel (File 705)
***Newport News Daily Press (File 747)
***U.S. Patents Fulltext 1980-1989 (File 653)

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
>>> of new databases, price changes, etc. <<<
****
* * * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.7.8 term=ASCII
*** DIALOG HOMEBASE(SM) Main Menu ***

Information:
1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:
6. DIALOG(R) Document Delivery
7. Data Star(R)

/H = Help /L = Logoff /NOMENU = Command Mode

Enter an option number to view information or to connect to an online
service. Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? b 410

22apr03 12:09:54 User268147 Session D67.1
$0.00 0.157 DialUnits FileHomeBase
$0.00 Estimated cost FileHomeBase
$0.00 Estimated cost this search
$0.00 Estimated total session cost 0.157 DialUnits

File 410:Chronolog(R) 1981-2003/Mar
(c) 2003 The Dialog Corporation

Set Items Description
--- ---- ---------
? set hi %%%;set hi %%%

```
HILIGHT set on as "
HILIGHT set on as "
? b 5, 34, 71, 155, 172, 434
     22apr03 12:10:12 User268147 Session D67.2
        $0.00   0.070 DialUnits File410
    $0.00  Estimated cost File410
    $0.06  TELNET
    $0.06  Estimated cost this search
    $0.06  Estimated total session cost   0.227 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2003/Apr W2
       (c) 2003 BIOSIS
*File   5: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  34:SciSearch(R) Cited Ref Sci  1990-2003/Apr W2
       (c) 2003 Inst for Sci Info
*File  34: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.
  File  71:ELSEVIER BIOBASE  1994-2003/Apr W3
       (c) 2003  Elsevier Science B.V.
  File 155:MEDLINE(R)  1966-2003/Apr W2
       (c) format only 2003 The Dialog Corp.
*File 155: Medline has been reloaded and accession numbers have
changed.  Please see HELP NEWS 155.
  File 172:EMBASE Alert  2003/Apr W3
       (c) 2003 Elsevier Science B.V.
  File 434:SciSearch(R) Cited Ref Sci  1974-1989/Dec
       (c) 1998 Inst for Sci Info

     Set  Items  Description
     ---  -----  ---------
? s "helicobacter pylori" or "h pylori"
         21056  HELICOBACTER PYLORI
            21  H PYLORI
     S1  21076  "HELICOBACTER PYLORI" OR "H PYLORI"
? s antisigma? or anti-sigma? or "anti sigma" or "anti sigma28"
            94  ANTISIGMA?
           216  ANTI-SIGMA?
             0  ANTI SIGMA
             0  ANTI SIGMA28
     S2    296  ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI
               SIGMA28"
? s jejuni or aeruginosa
         15102  JEJUNI
        101851  AERUGINOSA
     S3 116617  JEJUNI OR AERUGINOSA
? s s1 and (vector and "host cell")
         21076  S1
        298840  VECTOR
           197  HOST CELL
     S4      0  S1 AND (VECTOR AND "HOST CELL")
? s s4 and (vector? or "host cell" or "host cells")
             0  S4
        424167  VECTOR?
           197  HOST CELL
           194  HOST CELLS
     S5      0  S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
? ds

Set    Items  Description
S1     21076  "HELICOBACTER PYLORI" OR "H PYLORI"
```

```
S2      296  ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI SIGMA28"
S3   116617  JEJUNI OR AERUGINOSA
S4        0  S1 AND (VECTOR AND "HOST CELL")
S5        0  S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
? s s1 and recombinant
       21076 S1
      599594 RECOMBINANT
   S6    400 S1 AND RECOMBINANT
? e au=legrain pierre

Ref  Items Index-term
E1      32 AU=LEGRAIN P.
E2       1 AU=LEGRAIN PIERR
E3      37 *AU=LEGRAIN PIERRE
E4     152 AU=LEGRAIN R
E5       1 AU=LEGRAIN RL
E6      53 AU=LEGRAIN S
E7       8 AU=LEGRAIN S.
E8       3 AU=LEGRAIN SOPHIE
E9       8 AU=LEGRAIN SYLVIE
E10     59 AU=LEGRAIN V
E11      3 AU=LEGRAIN V.
E12      1 AU=LEGRAIN VALERIE

       Enter P or PAGE for more
? s e1 or e2 or e3
         32 AU=LEGRAIN P.
          1 AU=LEGRAIN PIERR
         37 AU=LEGRAIN PIERRE
   S7    70 AU='LEGRAIN P.' OR AU='LEGRAIN PIERR' OR AU='LEGRAIN
           PIERRE'
? e au=rain jean

Ref  Items Index-term
E1      12 AU=RAIN JC
E2     136 AU=RAIN JD
E3       0 *AU=RAIN JEAN
E4       1 AU=RAIN JEAN CHRISTOPHE
E5       1 AU=RAIN JEAN DIDIER
E6       9 AU=RAIN JEAN-CHRISTOPHE
E7      25 AU=RAIN JEAN-DIDIER
E8       1 AU=RAIN JJ
E9       1 AU=RAIN KH
E10      1 AU=RAIN L
E11      4 AU=RAIN M
E12      5 AU=RAIN M C

       Enter P or PAGE for more
? s e4 or e6
          1 AU=RAIN JEAN CHRISTOPHE
          9 AU=RAIN JEAN-CHRISTOPHE
   S8    10 AU='RAIN JEAN CHRISTOPHE' OR AU='RAIN JEAN-CHRISTOPHE'
? e au=de reuse

Ref  Items Index-term
E1       9 AU=DE REUS R
E2       1 AU=DE REUS-JORNA ANJA
E3       0 *AU=DE REUSE
E4      51 AU=DE REUSE H
E5      13 AU=DE REUSE H.
E6      19 AU=DE REUSE HILDE
E7       1 AU=DE REUTTER A S
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E8      4  AU=DE REUVER G F
E9      1  AU=DE REUVER J L
E10     1  AU=DE REUVER J.L.
E11     1  AU=DE REUVER LEO P
E12     1  AU=DE REUVER LP

       Enter P or PAGE for more
? s e4 or e5 or e6
         51  AU=DE REUSE H
         13  AU=DE REUSE H.
         19  AU=DE REUSE HILDE
   S9    83  AU='DE REUSE H' OR AU='DE REUSE H.' OR AU='DE REUSE
           HILDE'
? ds

Set    Items  Description
S1     21076  "HELICOBACTER PYLORI" OR "H PYLORI"
S2       296  ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI SIGMA28"
S3    116617  JEJUNI OR AERUGINOSA
S4         0  S1 AND (VECTOR AND "HOST CELL")
S5         0  S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
S6       400  S1 AND RECOMBINANT
S7        70  AU='LEGRAIN P.' OR AU='LEGRAIN PIERR' OR AU='LEGRAIN PIERR-
          E'
S8        10  AU='RAIN JEAN CHRISTOPHE' OR AU='RAIN JEAN-CHRISTOPHE'
S9        83  AU='DE REUSE H' OR AU='DE REUSE H.' OR AU='DE REUSE HILDE'
? s s7 or s8 or s9
         70  S7
         10  S8
         83  S9
   S10   147  S7 OR S8 OR S9
? ds

Set    Items  Description
S1     21076  "HELICOBACTER PYLORI" OR "H PYLORI"
S2       296  ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI SIGMA28"
S3    116617  JEJUNI OR AERUGINOSA
S4         0  S1 AND (VECTOR AND "HOST CELL")
S5         0  S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
S6       400  S1 AND RECOMBINANT
S7        70  AU='LEGRAIN P.' OR AU='LEGRAIN PIERR' OR AU='LEGRAIN PIERR-
          E'
S8        10  AU='RAIN JEAN CHRISTOPHE' OR AU='RAIN JEAN-CHRISTOPHE'
S9        83  AU='DE REUSE H' OR AU='DE REUSE H.' OR AU='DE REUSE HILDE'
S10      147  S7 OR S8 OR S9
? s s10 and s1
        147  S10
      21076  S1
   S11    20  S10 AND S1
? s py<=2001
Processing
Processing
Processing
Processing
Processing
   S1246731148  PY<=2001
? s s11 and s12
         20  S11
      46731148  S12
   S13    15  S11 AND S12
? type s13/full/all
```

13/9/1 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

10155806 Genuine Article#: 491RB Number of References: 19
Title: The Helicobacter pylori Urel protein is essential for survival to acidity and for the first steps of the mouse stomach colonization
Author(s): Bury-Mone S (REPRINT) ; Skouloubris S; Labigne A; De Reuse H
Corporate Source: Inst Pasteur,UPBM,28 Rue Docteur Roux/F-75724 Paris//France/ (REPRINT); Inst Pasteur,UPBM,F-75724 Paris//France/
Journal: GASTROENTEROLOGIE CLINIQUE ET BIOLOGIQUE, 2001, V25, N6-7 (JUN-JUL), P659-663
ISSN: 0399-8320 Publication date: 20010600
Publisher: MASSON EDITEUR, 120 BLVD SAINT-GERMAIN, 75280 PARIS 06, FRANCE
Language: French Document Type: ARTICLE
Geographic Location: France
Journal Subject Category: GASTROENTEROLOGY & HEPATOLOGY
Abstract: Helicobacter pylori (H. pylori) is a Gram negative microaerophilic bacteria whose only known niche is the human gastric mucosa. The presence of H. pylori is associated with various pathologies ranging from peptic ulcer disease to gastric carcinoma. H. pylori virulence is dependent on its exceptional ability to resist to the stomach acidity by hydrolyzing urea into ammonia. Survival of H. pylori to acidity in the presence of urea relies on the activity of a membrane protein, Urel.

Aims-We decided to better characterize the role of Urel (i) in vitro in ammonia production through the action of urease, and (ii) in vivo in the colonization of the gastric mucosa.

Methods-Ammonia production by a wild type strain of H. pylori or by a Urel-deficient strain was measured as a function of extracellular pH. In addition, the kinetics of elimination of a Urel-deficient mutant in vivo were realized in the mouse model for colonization.

Results-Urel was associated with an increase of ammonia production in acidic conditions in vitro and was necessary for the initial steps of the mouse stomach colonization.

Conclusion-Urel thus behaves as a sensor of extracellular pH. This protein activates urease at acidic pH,- thereby, it probably allows H. pylori to resist to acidity in vivo during the first steps of infection.
Descriptors--Author Keywords: Helicobacter pylori ; acidity ; urease ; ammonia ; Urel ; mouse model
Identifiers--KeyWord Plus(R): GNOTOBIOTIC PIGLETS; ALLELIC EXCHANGE; UREASE; GASTRITIS; INFECTION; STRAIN; MICE
Cited References:
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SKOULOUBRIS S, 1998, V66, P4517, INFECT IMMUN
TSUDA M, 1994, V62, P3586, INFECT IMMUN
WEEKS DL, 2000, V287, P482, SCIENCE
XU JK, 1990, V161, P1302, J INFECT DIS

13/9/2 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01817833 2001179768
Identification of the Helicobacter pylori anti-sigmaSUP28 factor
Colland F.; Rain J.-C.; Gounon P.; Labigne A.; Legrain P.; De Reuse H.
ADDRESS: H. De Reuse, U. Pathogenie Bacterienne Muqueuses, Institut Pasteur, 28 Rue du Dr Roux, 75724 Paris Cedex 15, France
EMAIL: hdereuse@pasteur.fr
Journal: Molecular Microbiology, 41/2 (477-487), 2001, United Kingdom
CODEN: MOMIE
ISSN: 0950-382X
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 52

Flagellar motility is essential for colonization of the human gastric mucosa by Helicobacter pylori. The flagellar filament is composed of two subunits, FlaA and FlaB. Transcription of the genes encoding these proteins is controlled by the sigmaSUP28 and sigmaSUP54 factors of RNA polymerase respectively. The expression of flagellar genes is regulated, but no sigmaSUP28-specific effector was identified. It was also unclear whether H. pylori possessed a checkpoint for flagellar synthesis, and no gene encoding an anti-sigmaSUP28 factor, FlgM, could be identified by sequence similarity searches. To investigate the sigmaSUP28-dependent regulation, a new approach based on genomic data was used. Two-hybrid screening with the H. pylori proteins identified a protein of unknown function (HP1122) interacting with the sigmaSUP28 factor and defined the C-terminal part of HP1122 (residues 48-76) as the interaction domain. HP1122 interacts with region 4 of sigmaSUP28 and prevents its association with the beta-region of H. pylori RNA polymerase. Thus, HP1122 presented the characteristics of an anti-sigmaSUP28 factor. This was confirmed in H. pylori by RNA dot-blot hybridization and electron microscopy. The level of sigmaSUP28-dependent flaA transcription was higher in a HP1122-deficient strain and was decreased by the overproduction of HP1122. The overproduction of HP1122 also resulted in H. pylori cells with highly truncated flagella. These results demonstrate that HP1122 is the H. pylori anti-sigmaSUP28 factor, FlgM, a major regulator of flagellum assembly. Potential anti-sigmaSUP28 factors were identified in Campylobacter jejuni, Pseudomonas aeruginosa and Thermotoga maritima by sequence homology with the C-terminal region of HP1122.

SPECIES DESCRIPTORS:
Helicobacter pylori; Campylobacter jejuni; Pseudomonas aeruginosa; Thermotoga maritima

CLASSIFICATION CODE AND DESCRIPTION:
85.12.1 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / METHODS AND TECHNIQUES / Identification and Characterization
85.7.17.4 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / MICROBIAL METABOLISM AND PHYSIOLOGY / Microbial Physiology / Chemotaxis and motility

13/9/3 (Item 2 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01743920 2001116911
The AmiE aliphatic amidase and AmiF formamidase of Helicobacter pylori: Natural evolution of two enzyme paralogues
Skouloubris S.; Labigne A.; De Reuse H.
ADDRESS: S. Skouloubris, Johns Hopkins University, Department of Chemistry, 3400 N. Charles St., Baltimore, MD 21218, United States
EMAIL: sskoulou@jhunix.hcf.jhu.edu
Journal: Molecular Microbiology, 40/3 (596-609), 2001, United Kingdom
CODEN: MOMIE
ISSN: 0950-382X
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 59

Aliphatic amidases (EC 3.5.1.4) are enzymes catalysing the hydrolysis of short-chain amides to produce ammonia and the corresponding organic acid. Such an amidase, AmiE, has been detected previously in Helicobacter pylori. Analysis of the complete H. pylori genome sequence revealed the existence of a duplicated amidase gene that we named amiF. The corresponding AmiF protein is 34% identical to its AmiE paralogue. Because gene duplication is widely considered to be a fundamental process in the acquisition of novel enzymatic functions, we decided to study and compare the functions of the paralogous amidases of H. pylori. AmiE and AmiF proteins were overproduced in Escherichia coli and purified by a two-step chromatographic procedure. The two H. pylori amidases could be distinguished by different biochemical characteristics such as optimum pH or temperature. AmiE hydrolysed propionamide, acetamide and acrylamide and had no activity with formamide. AmiF presented an unexpected substrate specificity: it only hydrolysed formamide. AmiF is thus the first formamidase (EC 3.5.1.49) related to aliphatic amidases to be described. Cys-165 in AmiE and Cys-166 in AmiF were identified as residues essential for catalysis of the corresponding enzymes. H. pylori strains carrying single and double mutations of amiE and amiF were constructed. The substrate specificities of these enzymes were confirmed in H. pylori. Production of AmiE and AmiF proteins is dependent on the activity of other enzymes involved in the nitrogen metabolism of H. pylori (urease and arginase respectively). Our results strongly suggest that (i) the H. pylori paralogous amidases have evolved to achieve enzymatic specialization after ancestral gene duplication; and (ii) the production of these enzymes is regulated to maintain intracellular nitrogen balance in H. pylori.

SPECIES DESCRIPTORS:
Helicobacter pylori

CLASSIFICATION CODE AND DESCRIPTION:
84.3.12 - GENETICS AND MOLECULAR BIOLOGY / PROKARYOTIC GENETICS / Prokaryotic Speciation and Evolution

MODLECULAR SEQUENCE DATABANK NUMBER:
UNNAMED/O25836/(REFERRED NUMBER)
UNNAMED/O32644/(REFERRED NUMBER)
UNNAMED/P11436/(REFERRED NUMBER)
UNNAMED/P32963/(REFERRED NUMBER)
UNNAMED/Q01360/(REFERRED NUMBER)
UNNAMED/Q03217/(REFERRED NUMBER)
UNNAMED/Q52445/(REFERRED NUMBER)

13/9/4 (Item 3 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

01668046 2001040222
The protein-protein interaction map of Helicobacter pylori
Rain J.-C.; Selig L.; De Reuse H.; Battaglia V.; Reverdy C.; Simon S.
; Lenzen G.; Petel F.; Wojcik J.; Schachter V.; Chemama Y.; Labigne A.;
Legrain P.
ADDRESS: P. Legrain, Hybrigenics SA, 180 avenue Daumesnil, Paris 75012,
France
EMAIL: plegrain@hybrigenics.fr
Journal: Nature, 409/6817 (211-215), 2001, United Kingdom
PUBLICATION DATE: January 11, 2001
CODEN: NATUA
ISSN: 0028-0836
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 23

With the availability of complete DNA sequences for many prokaryotic and eukaryotic genomes, and soon for the human genome itself, it is important to develop reliable proteome-wide approaches for a better understanding of protein functionSUP1. As elementary constituents of cellular protein complexes and pathways, protein-protein interactions are key determinants of protein function. Here we have built a large-scale protein-protein interaction map of the human gastric pathogen Helicobacter pylori. We have used a high-throughput strategy of the yeast two-hybrid assay to screen 261 H. pylori proteins against a highly complex library of genome-encoded polypeptidesSUP2. Over 1,200 interactions were identified between H. pylori proteins, connecting 46.6% of the proteome. The determination of a reliability score for every single protein-protein interaction and the identification of the actual interacting domains permitted the assignment of unannotated proteins to biological pathways.

SPECIES DESCRIPTORS:
Helicobacter pylori

CLASSIFICATION CODE AND DESCRIPTION:
82.2.12.2 - PROTEIN BIOCHEMISTRY / STRUCTURAL STUDIES / Molecular Recognition / Protein-protein interaction
82.12.7.3 - PROTEIN BIOCHEMISTRY / OTHER PROTEINS / Microbial Proteins / Bacterial
85.7.6 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / MICROBIAL METABOLISM AND PHYSIOLOGY / Nitrogen Transport and Metabolism
85.7.17.1 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / MICROBIAL METABOLISM AND PHYSIOLOGY / Microbial Physiology / Growth and differentiation

13/9/5 (Item 4 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE

00958171 1998204673
The Helicobacter pylori UreI protein is not involved in urease activity but is essential for bacterial survival in vivo
Skouloubris S.; Thiberge J.-M.; Labigne A.; De Reuse H.
ADDRESS: H. De Reuse, U. Pathogenie Bacterienne Muqueuses, Institut Pasteur, 28 rue du Dr. Roux, 75724 Paris Cedex 15, France
EMAIL: hdereuse@pasteur.fr
Journal: Infection and Immunity, 66/9 (4517-4521), 1998, United

States
CODEN: INFIB
ISSN: 0019-9567
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 25

We produced defined isogenic Helicobacter pylori ureI mutants to investigate the function of UreI, the product of one of the genes of the urease cluster. The insertion of a cat cassette had a strong polar effect on the expression of the downstream urease genes, resulting in very weak urease activity. Urease activity, measured in vitro, was normal in a strain in which ureI was almost completely deleted and replaced with a nonpolar cassette. In contrast to previous reports, we thus found that the product of ureI was not necessary for the synthesis of active urease. Experiments with the mouse- adapted H. pylori SS1 strain carrying the nonpolar ureI deletion showed that UreI is essential for H. pylori survival in vivo and/or colonization of the mouse stomach. The replacement of ureI with the nonpolar cassette strongly reduced H. pylori survival in acidic conditions (1-h incubation in phosphate- buffered saline solution at pH 2.2) in the presence of 10 mM urea. UreI is predicted to be an integral membrane protein and may therefore be involved in a transport process essential for H. pylori survival in vivo.

SPECIES DESCRIPTORS:
Helicobacter pylori

CLASSIFICATION CODE AND DESCRIPTION:
82.3.6 - PROTEIN BIOCHEMISTRY / PROTEIN ENGINEERING / Mutation, Expression and Isolation
82.3.7 - PROTEIN BIOCHEMISTRY / PROTEIN ENGINEERING / Mutant Proteins
82.12.7.3 - PROTEIN BIOCHEMISTRY / OTHER PROTEINS / Microbial Proteins / Bacterial
84.1.13.3 - GENETICS AND MOLECULAR BIOLOGY / MOLECULAR GENETICS / Molecular Biology Techniques / Gene targeting
84.3.6 - GENETICS AND MOLECULAR BIOLOGY / PROKARYOTIC GENETICS / Prokaryotic Biochemical Genetics
84.3.7 - GENETICS AND MOLECULAR BIOLOGY / PROKARYOTIC GENETICS / Genetics of Animal Pathogenesis

13/9/6 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11496180 98380408 PMID: 9712811
The Helicobacter pylori UreI protein is not involved in urease activity but is essential for bacterial survival in vivo.
Skouloubris S; Thiberge J M; Labigne A; De Reuse H
Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, 75724 Paris Cedex 15, France.
Infection and immunity (UNITED STATES) Sep 1998, 66 (9) p4517-21, ISSN 0019-9567 Journal Code: 0246127
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
We produced defined isogenic Helicobacter pylori ureI mutants to investigate the function of UreI, the product of one of the genes of the urease cluster. The insertion of a cat cassette had a strong polar effect on the expression of the downstream urease genes, resulting in very weak

urease activity. Urease activity, measured in vitro, was normal in a strain in which ureI was almost completely deleted and replaced with a nonpolar cassette. In contrast to previous reports, we thus found that the product of ureI was not necessary for the synthesis of active urease. Experiments with the mouse-adapted H. pylori SS1 strain carrying the nonpolar ureI deletion showed that UreI is essential for H. pylori survival in vivo and/or colonization of the mouse stomach. The replacement of ureI with the nonpolar cassette strongly reduced H. pylori survival in acidic conditions (1-h incubation in phosphate-buffered saline solution at pH 2.2) in the presence of 10 mM urea. UreI is predicted to be an integral membrane protein and may therefore be involved in a transport process essential for H. pylori survival in vivo.

Tags: Animal; Support, Non-U.S. Gov't

Descriptors: Bacterial Proteins--metabolism--ME; * Helicobacter pylori--growth and development--GD; *Helicobacter pylori --metabolism--ME; *Urease--metabolism--ME; Acids; Amino Acid Sequence; Bacterial Proteins--genetics--GE; Genes, Bacterial; Mice; Molecular Sequence Data; Mutagenesis; Sequence Homology, Amino Acid

CAS Registry No.: 0 (Acids); 0 (Bacterial Proteins); 0 (UreI protein)

Enzyme No.: EC 3.5.1.5 (Urease)

Record Date Created: 19981002

Record Date Completed: 19981002

13/9/7 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11154209 98030205 PMID: 9364923

Identification and characterization of an aliphatic amidase in Helicobacter pylori.

Skouloubris S; Labigne A; De Reuse H

Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, Paris, France.

Molecular microbiology (ENGLAND) Sep 1997, 25 (5) p989-98, ISSN 0950-382X Journal Code: 8712028

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

We report, for the first time, the presence in Helicobacter pylori of an aliphatic amidase that, like urease, contributes to ammonia production. Aliphatic amidases are cytoplasmic acylamide amidohydrolases (EC 3.5.1.4) hydrolysing short-chain aliphatic amides to produce ammonia and the corresponding organic acid. The finding of an aliphatic amidase in H. pylori was unexpected as this enzyme has only previously been described in bacteria of environmental (soil or water) origin. The H. pylori amidase gene amiE (1017 bp) was sequenced, and the deduced amino acid sequence of AmiE (37746Da) is very similar (75% identity) to the other two sequenced aliphatic amidases, one from Pseudomonas aeruginosa and one from Rhodococcus sp. R312. Amidase activity was measured as the release of ammonia by sonicated crude extracts from H. pylori strains and from recombinant Escherichia coli strains overproducing the H. pylori amidase. The substrate specificity was analysed with crude extracts from H. pylori cells grown in vitro; the best substrates were propionamide, acrylamide and acetamide. Polymerase chain reaction (PCR) amplification of an internal amiE sequence was obtained with each of 45 different H. pylori clinical isolates, suggesting that amidase is common to all H. pylori strains. A H. pylori mutant (N6-836) carrying an interrupted amiE gene was constructed by allelic exchange. No amidase activity could be detected in N6-836. In a

N6-urease negative mutant, amidase activity was two- to threefold higher than in the parental strain N6. Crude extracts of strain N6 slowly hydrolysed formamide. This activity was affected in neither the amidase negative strain (N6-836) nor a double mutant strain deficient in both amidase and urease activities, suggesting the presence of an independent discrete formamidase in H. pylori. The existence of an aliphatic amidase, a correlation between the urease and amidase activities and the possible presence of a formamidase indicates that H. pylori has a large range of possibilities for intracellular ammonia production.

Tags: Comparative Study; Support, Non-U.S. Gov't

Descriptors: Amidohydrolases--analysis--AN; *Helicobacter pylori --enzymology--EN; Amino Acid Sequence; Cloning, Molecular; DNA, Recombinant ; Escherichia coli--enzymology--EN; Escherichia coli--genetics--GE; Genes, Structural, Bacterial--genetics--GE; Helicobacter pylori--chemistry --CH; Helicobacter pylori--genetics--GE; Molecular Sequence Data; Mutation--genetics--GE; Recombinant Proteins--genetics--GE; Recombinant Proteins--metabolism--ME; Sequence Homology, Amino Acid; Substrate Specificity

Molecular Sequence Databank No.: GENBANK/Y12252

CAS Registry No.: 0 (DNA, Recombinant); 0 (Recombinant Proteins)

Enzyme No.: EC 3.5. (Amidohydrolases); EC 3.5.1.4 (amidase)

Record Date Created: 19980129

Record Date Completed: 19980129

13/9/8 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10962556 97315217 PMID: 9171391

The Helicobacter pylori ureC gene codes for a phosphoglucosamine mutase.

De Reuse H; Labigne A; Mengin-Lecreulx D

Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, Paris, France. hdereuse@pasteur.fr

Journal of bacteriology (UNITED STATES) Jun 1997, 179 (11) p3488-93, ISSN 0021-9193 Journal Code: 2985120R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The function of UreC, the product of a 1,335-bp-long open reading frame upstream from the urease structural genes (ureAB) of Helicobacter pylori, was investigated. We present data showing that the ureC gene product is a phosphoglucosamine mutase. D. Mengin-Lecreulx and J. van Heijenoort (J. Biol. Chem. 271:32-39, 1996) observed that UreC is similar (43% identity) to the GlmM protein of Escherichia coli. Those authors showed that GlmM is a phosphoglucosamine mutase catalyzing interconversion of glucosamine-6-phosphate into glucosamine-1-phosphate, which is subsequently transformed into UDP-N-acetylglucosamine. The latter product is one of the main cytoplasmic precursors of cell wall peptidoglycan and outer membrane lipopolysaccharides. The present paper reports that, like its E. coli homolog glmM, the H. pylori ureC gene is essential for cell growth. It was known that growth of a lethal conditional glmM mutant of E. coli at a nonpermissive temperature can be restored in the presence of the ureC gene. We showed that complete complementation of the glmM mutant can be obtained with a plasmid overproducing UreC. The peptidoglycan content and the specific phosphoglucosamine mutase activity of such a complemented strain were measured; these results demonstrated that the ureC gene product functions as a phosphoglucosamine mutase. Homologs of the UreC and GlmM proteins were identified in Haemophilus influenzae, Mycobacterium leprae, Clostridium perfringens, Synechocystis sp. strain PCC6803, and

Methanococcus jannaschii. Significant conservation of the amino acid sequence of these proteins in such diverse organisms suggests a very ancient common ancestor for the genes and defines a consensus motif for the phosphoglucosamine mutase active site. We propose renaming the H. pylori ureC gene the glmM gene.
Descriptors: Genes, Bacterial; *Helicobacter pylori--genetics--GE; *Phosphoglucomutase--genetics--GE; Amino Acid Sequence; Escherichia coli --genetics--GE; Helicobacter pylori--enzymology--EN; Molecular Sequence Data; Sequence Alignment
Enzyme No.: EC 5.4.2.- (phosphoglucosamine mutase); EC 5.4.2.2 (Phosphoglucomutase)
Record Date Created: 19970703
Record Date Completed: 19970703

13/9/9 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10689580 97038783 PMID: 8884364
Determinants of Helicobacter pylori pathogenicity.
Labigne A; de Reuse H
Unite de Pathogenie Bacterienne des Muqueuses, Unite INSERM U389, Institut Pasteur, Paris, France.
Infectious agents and disease (UNITED STATES) Oct 1996, 5 (4) p191-202, ISSN 1056-2044 Journal Code: 9209834
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Helicobacter pylori is a recently recognized bacterial pathogen associated with diverse pathologies of varying severity, such as chronic gastritis, peptic ulceration, mucosa-associated lymphoid tissue (MALT) lymphoma, and gastric carcinoma. We here present a review of our current knowledge on the properties of H. Pylori that adapt it to its particular niche by allowing it to survive in the stomach and to colonize the gastric mucosa, as well as those that underlie its persistence and pathogenicity. While the bacterial determinants that preclude the persistent colonization of the gastric mucosa are better understood, those associated with pathogenicity appear to result from the possibility for some of the bacteria of the species to synthesize products that directly or indirectly damage the gastric mucosa, cause a persistent inflammatory reaction, and/or perturb the regulation of acid secretion. (110 Refs.)
Tags: Human
Descriptors: Gastric Mucosa--metabolism--ME; *Gastric Mucosa --microbiology--MI; *Helicobacter pylori--pathogenicity--PY; Bacterial Adhesion; Gastric Acid--secretion--SE; Helicobacter Infections --epidemiology--EP
Record Date Created: 19970121
Record Date Completed: 19970121

13/9/10 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09795535 21602923 PMID: 11737644
The Helicobacter pylori UreI protein: role in adaptation to acidity and identification of residues essential for its activity and for acid activation.
Bury-Mone S; Skouloubris S; Labigne A; De Reuse H

Institut Pasteur, Unite de Pathogenie Bacterienne des Muqueuses, 28 rue du Dr Roux, 75724 Paris Cedex 15, France.
Molecular microbiology (England) Nov 2001, 42 (4) p1021-34, ISSN 0950-382X Journal Code: 8712028
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori is a human gastric pathogen that survives the strong acidity of the stomach by virtue of its urease activity. This activity produces ammonia, which neutralizes the bacterial microenvironment. UreI, an inner membrane protein, is essential for resistance to low pH and for the gastric colonization of mice by H. pylori. In the heterologous Xenopus oocytes expression system, UreI behaves like an H+-gated urea channel, and His-123 was found to be important for low pH activation. We investigated the role of UreI directly in H. pylori and showed that, in the presence of urea, strains expressing wild-type UreI displayed very rapid stimulation of extracellular ammonia production upon exposure to pH </= 5. This response was not observed when acetamide was used as a source of ammonia; therefore, it is specific for urea hydrolysis. To identify residues critical for UreI activity or activation, we constructed H. pylori strains carrying individual chromosomal mutations of UreI (i) in the four conserved histidine residues (H71, H123, H131, H193) and (ii) in a conserved region of the third intracellular loop (L165, G166, K167, F168). The distal H193 (and not H123) was found to be crucial for stimulating the production of ammonia at low pH; a single mutation in this residue uncoupled the UreI activity from its acid activation. The third intracellular loop of UreI was shown to be important for UreI activity. Thus, in H. pylori, UreI is necessary for the adaptation of urease activity to the extracellular pH. UreI behaves like a novel type of urea transporter, and the identification of residues essential for its function in H. pylori provides new insight into the unusual molecular mechanism of low pH activation.

Tags: Animal; Human
Descriptors: Bacterial Proteins--metabolism--ME; * Helicobacter pylori--physiology--PH; Acetamides--pharmacology--PD; Adaptation, Physiological; Amino Acid Sequence; Ammonia--metabolism--ME; Bacterial Proteins--chemistry--CH; Bacterial Proteins--genetics--GE; Chimeric Proteins--metabolism--ME; Genes, Bacterial; Helicobacter pylori --cytology--CY; Helicobacter pylori--drug effects--DE; Helicobacter pylori--genetics--GE; Hydrogen-Ion Concentration; Hydrolysis; Molecular Sequence Data; Mutagenesis, Site-Directed; Phenotype; Protein Structure, Secondary; Sequence Alignment; Urea--metabolism--ME; Urea--pharmacology--PD; Urease--metabolism--ME
CAS Registry No.: 0 (Acetamides); 0 (Bacterial Proteins); 0 (Chimeric Proteins); 0 (UreI protein); 57-13-6 (Urea); 60-35-5 (acetamide); 7664-41-7 (Ammonia)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 20011212
Record Date Completed: 20020301

13/9/11 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09729682 21526966 PMID: 11673732
[UreI: a Helicobacter pylori protein essential for resistance to acidity and for the early steps of murine gastric mucosa infection]
UreI: une proteine de Helicobacter pylori essentielle a la resistance a l'acidite et aux etapes precoces de l'infection de la muqueuse gastrique murine.

Bury-Mone S; Skouloubris S; Labigne A; De Reuse H
Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, Paris. sbury@pasteur.fr
Gastroenterologie clinique et biologique (France) Jun-Jul 2001, 25 (6-7) p659-63, ISSN 0399-8320 Journal Code: 7704825
Document type: Journal Article ; English Abstract
Languages: FRENCH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Helicobacter pylori (H. pylori) is a Gram negative microaerophilic bacteria whose only known niche is the human gastric mucosa. The presence of H. pylori is associated with various pathologies ranging from peptic ulcer disease to gastric carcinoma. H. pylori virulence is dependent on its exceptional ability to resist to the stomach acidity by hydrolyzing urea into ammonia. Survival of H. pylori to acidity in the presence of urea relies on the activity of a membrane protein, UreI.AIMS: We decided to better characterize the role of UreI (i) in vitro in ammonia production through the action of urease, and (ii) in vivo in the colonization of the gastric mucosa.METHODS: Ammonia production by a wild type strain of H. pylori or by a UreI-deficient strain was measured as a function of extracellular pH. In addition, the kinetics of elimination of a UreI-deficient mutant in vivo were realized in the mouse model for colonization.RESULTS: UreI was associated with an increase of ammonia production in acidic conditions in vitro and was necessary for the initial steps of the mouse stomach colonization.CONCLUSION: UreI thus behaves as a sensor of extracellular pH. This protein activates urease at acidic pH; thereby, it probably allows H. pylori to resist to acidity in vivo during the first steps of infection.

Tags: Animal
Descriptors: Bacterial Proteins--physiology--PH; *Disease Models, Animal; *Gastric Acid--physiology--PH; *Gastric Mucosa--microbiology--MI; *Helicobacter Infections--microbiology--MI; *Helicobacter pylori --pathogenicity--PY; *Helicobacter pylori--physiology--PH; *Stomach Diseases--microbiology--MI; Ammonia--metabolism--ME; Colony Count, Microbial; Hydrogen-Ion Concentration; Hydrolysis; Mice; Time Factors; Urea --metabolism--ME
CAS Registry No.: 0 (Bacterial Proteins); 0 (UreI protein); 57-13-6 (Urea); 7664-41-7 (Ammonia)
Record Date Created: 20011023
Record Date Completed: 20011205

13/9/12 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09598550 21382773 PMID: 11489132
Identification of the Helicobacter pylori anti-sigma28 factor.
Colland F; Rain J C; Gounon P; Labigne A; Legrain P; De Reuse H
Hybrigenics SA, 180 avenue Daumesnil, Paris 75012, France.
Molecular microbiology (England) Jul 2001, 41 (2) p477-87, ISSN 0950-382X Journal Code: 8712028
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

Flagellar motility is essential for colonization of the human gastric mucosa by Helicobacter pylori. The flagellar filament is composed of two subunits, FlaA and FlaB. Transcription of the genes encoding these proteins is controlled by the sigma28 and sigma54 factors of RNA polymerase

respectively. The expression of flagellar genes is regulated, but no sigma28-specific effector was identified. It was also unclear whether H. pylori possessed a checkpoint for flagellar synthesis, and no gene encoding an anti-sigma28 factor, FlgM, could be identified by sequence similarity searches. To investigate the sigma28-dependent regulation, a new approach based on genomic data was used. Two-hybrid screening with the H. pylori proteins identified a protein of unknown function (HP1122) interacting with the sigma28 factor and defined the C-terminal part of HP1122 (residues 48-76) as the interaction domain. HP1122 interacts with region 4 of sigma28 and prevents its association with the beta-region of H. pylori RNA polymerase. Thus, HP1122 presented the characteristics of an anti-sigma28 factor. This was confirmed in H. pylori by RNA dot-blot hybridization and electron microscopy. The level of sigma28-dependent flaA transcription was higher in a HP1122-deficient strain and was decreased by the overproduction of HP1122. The overproduction of HP1122 also resulted in H. pylori cells with highly truncated flagella. These results demonstrate that HP1122 is the H. pylori anti-sigma28 factor, FlgM, a major regulator of flagellum assembly. Potential anti-sigma28 factors were identified in Campylobacter jejuni, Pseudomonas aeruginosa and Thermotoga maritima by sequence homology with the C-terminal region of HP1122.

Descriptors: Bacterial Proteins--antagonists and inhibitors--AI; *Bacterial Proteins--metabolism--ME; *Gene Expression Regulation, Bacterial; *Genes, Bacterial--genetics--GE; *Helicobacter pylori --genetics--GE; *Sigma Factor--antagonists and inhibitors--AI; Amino Acid Sequence; Bacterial Proteins--chemistry--CH; Bacterial Proteins--genetics --GE; Binding Sites; Cloning, Molecular; DNA-Directed RNA Polymerases --chemistry--CH; DNA-Directed RNA Polymerases--metabolism--ME; Flagella --metabolism--ME; Flagella--ultrastructure--UL; Flagellin--genetics--GE; Gene Deletion; Helicobacter pylori--cytology--CY; Helicobacter pylori--metabolism--ME; Helicobacter pylori--ultrastructure--UL; Molecular Sequence Data; Promoter Regions (Genetics)--genetics--GE; Protein Binding; Protein Structure, Tertiary; RNA, Bacterial--genetics--GE; RNA, Bacterial--metabolism--ME; Sequence Homology, Amino Acid; Sigma Factor --metabolism--ME; Transcription, Genetic; Two-Hybrid System Techniques

CAS Registry No.: 0 (Bacterial Proteins); 0 (FliA protein); 0 (RNA, Bacterial); 0 (Sigma Factor); 12777-81-0 (Flagellin); 133606-66-3 (flaA protein); 142462-45-1 (FlgM protein)

Enzyme No.: EC 2.7.7.6 (DNA-Directed RNA Polymerases)

Record Date Created: 20010807

Record Date Completed: 20020411

13/9/13 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09483839 21260068 PMID: 11359566

The AmiE aliphatic amidase and AmiF formamidase of Helicobacter pylori: natural evolution of two enzyme paralogues.

Skouloubris S; Labigne A; De Reuse H

Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, 28 rue du Docteur Roux, 75724 Paris Cedex 15, France.sskoulou@jhunix.hcf.jhu.edu

Molecular microbiology (England) May 2001, 40 (3) p596-609, ISSN 0950-382X Journal Code: 8712028

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Aliphatic amidases (EC 3.5.1.4) are enzymes catalysing the hydrolysis of short-chain amides to produce ammonia and the corresponding organic acid. Such an amidase, AmiE, has been detected previously in Helicobacter pylori.

Analysis of the complete H. pylori genome sequence revealed the existence of a duplicated amidase gene that we named amiF. The corresponding AmiF protein is 34% identical to its AmiE paralogue. Because gene duplication is widely considered to be a fundamental process in the acquisition of novel enzymatic functions, we decided to study and compare the functions of the paralogous amidases of H. pylori. AmiE and AmiF proteins were overproduced in Escherichia coli and purified by a two-step chromatographic procedure. The two H. pylori amidases could be distinguished by different biochemical characteristics such as optimum pH or temperature. AmiE hydrolysed propionamide, acetamide and acrylamide and had no activity with formamide. AmiF presented an unexpected substrate specificity: it only hydrolysed formamide. AmiF is thus the first formamidase (EC 3.5.1.49) related to aliphatic amidases to be described. Cys-165 in AmiE and Cys-166 in AmiF were identified as residues essential for catalysis of the corresponding enzymes. H. pylori strains carrying single and double mutations of amiE and amiF were constructed. The substrate specificities of these enzymes were confirmed in H. pylori. Production of AmiE and AmiF proteins is dependent on the activity of other enzymes involved in the nitrogen metabolism of H. pylori (urease and arginase respectively). Our results strongly suggest that (i) the H. pylori paralogous amidases have evolved to achieve enzymatic specialization after ancestral gene duplication; and (ii) the production of these enzymes is regulated to maintain intracellular nitrogen balance in H. pylori.

Tags: Support, Non-U.S. Gov't

Descriptors: Amidohydrolases--genetics--GE; *Evolution, Molecular; * Helicobacter pylori--enzymology--EN; Amidohydrolases--isolation and purification--IP; Amidohydrolases--metabolism--ME; Amino Acid Sequence; Cloning, Molecular; Escherichia coli; Gene Expression; Genes, Bacterial; Helicobacter pylori--genetics--GE; Molecular Sequence Data; Mutagenesis, Site-Directed; Sequence Homology, Amino Acid

Enzyme No.: EC 3.5. (Amidohydrolases); EC 3.5.1.4 (amidase); EC 3.5.1.49 (formamidase)

Record Date Created: 20010521

Record Date Completed: 20010927

13/9/14 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09300238 21037968 PMID: 11196647

The protein-protein interaction map of Helicobacter pylori.

Rain J C; Selig L; De Reuse H; Battaglia V; Reverdy C; Simon S; Lenzen G; Petel F; Wojcik J; Schachter V; Chemama Y; Labigne A; Legrain P

Hybrigenics SA, Paris, France.

Nature (England) Jan 11 2001, 409 (6817) p211-5, ISSN 0028-0836 Journal Code: 0410462

Erratum in Nature 2001 Feb 1;409(6820) 553; Erratum in Nature 2001 Feb 8;409(6821):743

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

With the availability of complete DNA sequences for many prokaryotic and eukaryotic genomes, and soon for the human genome itself, it is important to develop reliable proteome-wide approaches for a better understanding of protein function. As elementary constituents of cellular protein complexes and pathways, protein-protein interactions are key determinants of protein function. Here we have built a large-scale protein-protein interaction map of the human gastric pathogen Helicobacter pylori. We have used a high-throughput strategy of the yeast two-hybrid assay to screen 261 H.

pylori proteins against a highly complex library of genome-encoded polypeptides. Over 1,200 interactions were identified between H. pylori proteins, connecting 46.6% of the proteome. The determination of a reliability score for every single protein-protein interaction and the identification of the actual interacting domains permitted the assignment of unannotated proteins to biological pathways.
Tags: Human; Support, Non-U.S. Gov't
Descriptors: Bacterial Proteins--metabolism--ME; * Helicobacter pylori--metabolism--ME; Amino Acid Sequence; Binding Sites; Databases, Factual; Escherichia coli--genetics--GE; Gene Library; Internet; Molecular Sequence Data; Protein Binding; Proteome; Sequence Alignment; Software; Urease--metabolism--ME
CAS Registry No.: 0 (Bacterial Proteins); 0 (Proteome)
Enzyme No.: EC 3.5.1.5 (Urease)
Record Date Created: 20010122
Record Date Completed: 20010201

13/9/15 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09167782 20471137 PMID: 11019630
[Bacteriology and pathogenicity of Helicobacter pylori]
Bacteriologie et pathogenicite d'Helicobacter pylori.
Skouloubris S; De Reuse H; Labigne A
Unite de pathogenie bacterienne des muqueuses Institut Pasteur, Paris. sskoulou@pasteur.fr
La Revue du praticien (FRANCE) Sep 1 2000, 50 (13) p1409-13, ISSN 0035-2640 Journal Code: 0404334
Document type: Journal Article ; English Abstract
Languages: FRENCH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Helicobacter pylori is the prototype of bacteria belonging to a new genus, the Helicobacter genus. It is a gram-negative, highly motile and microaerophilic bacterium, with a spiral shape, that colonizes the human gastric mucosa and causes several gastroduodenal diseases. Pathogenicity of H. pylori relies upon its capacity to adapt to a hostile environment and to escape the host response. Resistance to acidity, motility, adhesion, molecular mimicry, resistance to phagocytosis, synthesis of a cytotoxin, induction of an inflammatory response are the major strategies developed by H. pylori to colonize persistently and damage gastric tissue.
Tags: Human
Descriptors: Helicobacter Infections--physiopathology--PP; * Helicobacter pylori; *Stomach--physiology--PH; Cell Survival; Cytotoxins--biosynthesis--BI; Gastric Mucosa--microbiology--MI; Gastric Mucosa--pathology--PA; Hydrogen-Ion Concentration; Phagocytosis
CAS Registry No.: 0 (Cytotoxins)
Record Date Created: 20001208
Record Date Completed: 20001208
? ds

Set	Items	Description
S1	21076	"HELICOBACTER PYLORI" OR "H PYLORI"
S2	296	ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI SIGMA28"
S3	116617	JEJUNI OR AERUGINOSA
S4	0	S1 AND (VECTOR AND "HOST CELL")
S5	0	S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
S6	400	S1 AND RECOMBINANT
S7	70	AU='LEGRAIN P.' OR AU='LEGRAIN PIERR' OR AU='LEGRAIN PIERR-

E'
S8 10 AU='RAIN JEAN CHRISTOPHE' OR AU='RAIN JEAN-CHRISTOPHE'
S9 83 AU='DE REUSE H' OR AU='DE REUSE H.' OR AU='DE REUSE HILDE'
S10 147 S7 OR S8 OR S9
S11 20 S10 AND S1
S12 46731148 PY<=2001
S13 15 S11 AND S12
? s s s2 and s6
0 S S2
400 S6
S14 0 S S2 AND S6
? s s1 and s2
21076 S1
296 S2
S15 0 S1 AND S2
? s pylori? and s2
98221 PYLORI?
296 S2
S16 5 PYLORI? AND S2
? type s16/full/all

16/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13442256 BIOSIS NO.: 200200071077
Identification of the flagellar type III system anti-sigma factor FlgM of Helicobacter pylori and characterisation of FlgM and FliA mutants.
AUTHOR: Josenhans C V(a); Amersbach S(a); Stack A(a); Niehus E(a); Suerbaum S(a)
AUTHOR ADDRESS: (a)Wuerzburg University, Wuerzburg**Germany
JOURNAL: IJMM International Journal of Medical Microbiology 291 (Supplement 31):p24 September, 2001
MEDIUM: print
CONFERENCE/MEETING: 11th International Workshop on Campylobacter, Helicobacter and related Organisms Freiburg, Germany September 01-05, 2001
ISSN: 1438-4221
RECORD TYPE: Citation
LANGUAGE: English
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Infection; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Aerobic Helical or Vibrioid Gram-Negatives--Eubacteria, Bacteria, Microorganisms; Enterobacteriaceae--Facultatively Anaerobic Gram-Negative Rods, Eubacteria, Bacteria, Microorganisms; Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Helicobacter pylori (Aerobic Helical or Vibrioid Gram-Negatives)--pathogen; Salmonella typhimurium (Enterobacteriaceae) --pathogen; human (Hominidae)--host
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals; Microorganisms; Primates; Vertebrates
CHEMICALS & BIOCHEMICALS: anti-sigma28 factor; flagellin; flgM; flhCD flagellar operon; fliA
GENE NAME: Helicobacter pylori flgM gene (Aerobic Helical or Vibrioid Gram-Negatives)
METHODS & EQUIPMENT: western blot--analytical method, gene mapping, labeling
MISCELLANEOUS TERMS: Meeting Abstract
CONCEPT CODES:
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals

02502 Cytology and Cytochemistry-General
02508 Cytology and Cytochemistry-Human
03502 Genetics and Cytogenetics-General
03508 Genetics and Cytogenetics-Human
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
30500 Morphology and Cytology of Bacteria
31000 Physiology and Biochemistry of Bacteria
31500 Genetics of Bacteria and Viruses
BIOSYSTEMATIC CODES:
06210 Aerobic Helical or Vibrioid Gram-Negatives (1992-)
06702 Enterobacteriaceae (1992-)
86215 Hominidae

16/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13442255 BIOSIS NO.: 200200071076
Regulation of flagellar assembly in Helicobacter pylori:
Identification of the anti-sigma28 factor (HP1122).
AUTHOR: Colland F(a); Rain J(a); Gounon P; Labigne A; Legrain P(a); De Reuse H
AUTHOR ADDRESS: (a)Hybrigenics SA, Paris**France
JOURNAL: IJMM International Journal of Medical Microbiology 291 (Supplement 31):p23 September, 2001
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DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Infection; Molecular Genetics (Biochemistry and Molecular Biophysics); Movement and Support
BIOSYSTEMATIC NAMES: Aerobic Helical or Vibrioid Gram-Negatives--Eubacteria, Bacteria, Microorganisms; Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Pseudomonadaceae--Gram-Negative Aerobic Rods and Cocci, Eubacteria, Bacteria, Microorganisms
ORGANISMS: Campylobacter jejuni (Aerobic Helical or Vibrioid Gram-Negatives)--pathogen; Helicobacter pylori (Aerobic Helical or Vibrioid Gram-Negatives)--pathogen; Pseudomonas aeruginosa (Pseudomonadaceae)--pathogen; human (Hominidae)--host
ORGANISMS: PARTS ETC: flagellum
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Bacteria; Chordates; Eubacteria; Humans; Mammals; Microorganisms; Primates; Vertebrates
CHEMICALS & BIOCHEMICALS: FlaA; FlaB; RNA; RNA polymerase; anti-sigma28 factor {HP1122}
MISCELLANEOUS TERMS: flagellar assembly; motility; Meeting Abstract
CONCEPT CODES:
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
02502 Cytology and Cytochemistry-General
02508 Cytology and Cytochemistry-Human
03502 Genetics and Cytogenetics-General
03508 Genetics and Cytogenetics-Human
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10802 Enzymes-General and Comparative Studies; Coenzymes
12002 Physiology, General and Miscellaneous-General

30500 Morphology and Cytology of Bacteria
31000 Physiology and Biochemistry of Bacteria
31500 Genetics of Bacteria and Viruses
BIOSYSTEMATIC CODES:
06210 Aerobic Helical or Vibrioid Gram-Negatives (1992-)
06508 Pseudomonadaceae (1992-)
86215 Hominidae

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DIALOG(R)File 5:Biosis Previews(R)

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Identification of the Helicobacter pylori anti-sigma28 factor.
AUTHOR: Colland Frederic; Rain Jean-Christophe; Gounon Pierre; Labigne Agnes; Legrain Pierre; De Reuse Hilde(a)
AUTHOR ADDRESS: (a)Unite de Pathogenie Bacterienne des Muqueuses, Institut Pasteur, 28 Rue du Dr Roux, 75724, Paris Cedex 15: hdereuse@pasteur.fr** France
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SUMMARY LANGUAGE: English

ABSTRACT: Flagellar motility is essential for colonization of the human gastric mucosa by Helicobacter pylori. The flagellar filament is composed of two subunits, FlaA and FlaB. Transcription of the genes encoding these proteins is controlled by the sigma28 and sigma54 factors of RNA polymerase respectively. The expression of flagellar genes is regulated, but no sigma28-specific effector was identified. It was also unclear whether H. pylori possessed a checkpoint for flagellar synthesis, and no gene encoding an anti-sigma28 factor, FlgM, could be identified by sequence similarity searches. To investigate the sigma28-dependent regulation, a new approach based on genomic data was used. Two-hybrid screening with the H. pylori proteins identified a protein of unknown function (HP1122) interacting with the sigma28 factor and defined the C-terminal part of HP1122 (residues 48-76) as the interaction domain. HP1122 interacts with region 4 of sigma28 and prevents its association with the beta-region of H. pylori RNA polymerase. Thus, HP1122 presented the characteristics of an anti-sigma28 factor. This was confirmed in H. pylori by RNA dot-blot hybridization and electron microscopy. The level of sigma28-dependent flaA transcription was higher in a HP1122-deficient strain and was decreased by the overproduction of HP1122. The overproduction of HP1122 also resulted in H. pylori cells with highly truncated flagella. These results demonstrate that HP1122 is the H. pylori anti-sigma28 factor, FlgM, a major regulator of flagellum assembly. Potential anti-sigma28 factors were identified in Campylobacter jejuni, Pseudomonas aeruginosa and Thermotoga maritima by sequence homology with the C-terminal region of HP1122.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics
BIOSYSTEMATIC NAMES: Aerobic Helical or Vibrioid Gram-Negatives--Eubacteria, Bacteria, Microorganisms
ORGANISMS: Helicobacter pylori (Aerobic Helical or Vibrioid Gram-Negatives)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Bacteria; Eubacteria;

Microorganisms
CHEMICALS & BIOCHEMICALS: FlgM--anti-sigma 28 factor, identification
MISCELLANEOUS TERMS: flagellar motility
CONCEPT CODES:
10060 Biochemical Studies-General
31000 Physiology and Biochemistry of Bacteria
BIOSYSTEMATIC CODES:
06210 Aerobic Helical or Vibrioid Gram-Negatives (1992-)

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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09934069 Genuine Article#: 465AU Number of References: 52
Title: Identification of the Helicobacter pylori anti-sigma(28) factor
Author(s): Colland F; Rain JC; Gounon P; Labigne A; Legrain P; De Reuse H (REPRINT)
Corporate Source: Inst Pasteur,Unite Pathogenie Bacterienne Muqueuses,28 Rue Dr Roux/F-75724 Paris 15//France/ (REPRINT); Inst Pasteur,Unite Pathogenie Bacterienne Muqueuses,F-75724 Paris 15//France/; Hybrigenics SA,F-75012 Paris//France/; Inst Pasteur,Stn Cent Microscopie Elect,F-75724 Paris 15//France/
Journal: MOLECULAR MICROBIOLOGY, 2001, V41, N2 (JUL), P477-487
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Publisher: BLACKWELL SCIENCE LTD, P O BOX 88, OSNEY MEAD, OXFORD OX2 0NE, OXON, ENGLAND
Language: English Document Type: ARTICLE
Geographic Location: France
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; MICROBIOLOGY
Abstract: Flagellar motility is essential for colonization of the human gastric mucosa by Helicobacter pylori. The flagellar filament is composed of two subunits, FlaA and FlaB. Transcription of the genes encoding these proteins is controlled by the sigma (28) and sigma (54) factors of RNA polymerase respectively. The expression of flagellar genes is regulated, but no sigma (28)-specific effector was identified. It was also unclear whether H. pylori possessed a checkpoint for flagellar synthesis, and no gene encoding an anti-sigma (28) factor, FIgM, could be identified by sequence similarity searches. To investigate the sigma (28)-dependent regulation, a new approach based on genomic data was used. Two-hybrid screening with the H. pylori proteins identified a protein of unknown function (HP1122 interacting with the sigma (28) factor and defined the C-terminal part of HP1122 (residues 48-76) as the interaction domain. HP1122 interacts with region 4 of sigma (28) and prevents its association with the beta -region of H. pylori RNA polymerase. Thus, HP1122 presented the characteristics of an anti-sigma (28) factor. This was confirmed in H. pylori by RNA dot-blot hybridization and electron microscopy. The level of sigma (28)-dependent flaA transcription was higher in a HP1122-deficient strain and was decreased by the overproduction of HP1122. The overproduction of HP1122 also resulted in H. pylori cells with highly truncated flagella. These results demonstrate that HP1122 is the H. pylori anti-sigma (28) factor, FIgM, a major regulator of flagellum assembly. Potential anti-sigma (28) factors were identified in Campylobacter jejuni, Pseudomonas aeruginosa and Thermotoga maritima by sequence homology with the C-terminal region of HP1122.
Identifiers--KeyWord Plus(R): ANTI-SIGMA FACTOR; FLAGELLUM-SPECIFIC SIGMA; COMPLETE GENOME SEQUENCE; SALMONELLA-TYPHIMURIUM; TRANSCRIPTIONAL REGULATION; CAMPYLOBACTER-JEJUNI;

GENETIC-CHARACTERIZATION; ESCHERICHIA-COLI; NEGATIVE MUTANTS; ALLELIC EXCHANGE

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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08214036 Genuine Article#: 257PG Number of References: 40

Title: Molecular cloning and characterization of the Helicobacter pylori fliD gene, an essential factor in flagellar structure and motility

Author(s): Kim JS; Chang JH; Chung SI; Yum JS (REPRINT)
Corporate Source: MOGAM BIOTECHNOL RES INST,341 POJUNG RI/YONGIN 449910/KYONGGI DO/SOUTH KOREA/ (REPRINT); MOGAM BIOTECHNOL RES INST,/YONGIN 449910/KYONGGI DO/SOUTH KOREA/

Journal: JOURNAL OF BACTERIOLOGY, 1999, V181, N22 (NOV), P6969-6976
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Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: MICROBIOLOGY

Abstract: Helicobacter pylori colonizes the human stomach and can cause gastroduodenal disease. Flagellar motility is regarded as a major factor in the colonizing ability of H. pylori, The functional roles of flagellar structural proteins other than FlaA, FlaB, and FlgE are not well understood. The fliD operon of H. pylori consists of flaG, fliD, and fliS genes, in the order stated, under the control of a sigma(28)-dependent promoter. In an effort to elucidate the function of the FliD protein, a hook-associated protein 2 homologue, in flagellar morphogenesis and motility, the fliD gene (2,058 bp) was cloned and isogenic mutants were constructed by disruption of the fliD gene with a kanamycin resistance cassette and electroporation-mediated allelic-exchange mutagenesis. In the fliD mutant, morphologically abnormal flagellar appendages in which very little filament elongation was apparent were observed. The fliD mutant strain was completely nonmotile, indicating that these abnormal flagella were functionally defective. Furthermore, the isogenic fliD mutant of H. pylori SS1, a mouse-adapted strain, was not able to colonize the gastric mucosae of host mice. These results suggest that H. pylori FliD is an essential element in the assembly of the functional flagella that are required for colonization of the gastric mucosa.

Identifiers--KeyWord Plus(R): HOOK-ASSOCIATED PROTEINS; ANTI-SIGMA FACTOR; SALMONELLA-TYPHIMURIUM; NEGATIVE MUTANTS; ALLELIC EXCHANGE; MOUSE MODEL; CONSTRUCTION; INFECTION; REGULON; INTERNALIZATION

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Set	Items	Description
S1	21076	"HELICOBACTER PYLORI" OR "H PYLORI"
S2	296	ANTISIGMA? OR ANTI-SIGMA? OR "ANTI SIGMA" OR "ANTI SIGMA28"
S3	116617	JEJUNI OR AERUGINOSA
S4	0	S1 AND (VECTOR AND "HOST CELL")
S5	0	S4 AND (VECTOR? OR "HOST CELL" OR "HOST CELLS")
S6	400	S1 AND RECOMBINANT
S7	70	AU='LEGRAIN P.' OR AU='LEGRAIN PIERR' OR AU='LEGRAIN PIERR-E'
S8	10	AU='RAIN JEAN CHRISTOPHE' OR AU='RAIN JEAN-CHRISTOPHE'
S9	83	AU='DE REUSE H' OR AU='DE REUSE H.' OR AU='DE REUSE HILDE'
S10	147	S7 OR S8 OR S9
S11	20	S10 AND S1
S12	46731148	PY<=2001
S13	15	S11 AND S12
S14	0	S S2 AND S6
S15	0	S1 AND S2
S16	5	PYLORI? AND S2